A Dissertation on

A STUDY ON CONDUCTION ABNORMALITIES AND QRS DURATION IN PATIENTS WITH ISCHEMIC CARDIOMYOPATHY AND THEIR CORRELATION WITH EJECTION FRACTION

submitted to

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

CHENNAI

In partial fulfillment of the regulations

for the award of the degree of

M.D.BRANCH - I

GENERAL MEDICINE

GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL

CHENNAI – TAMIL NADU

APRIL 2012

CERTIFICATE

This is to certify that this dissertation entitled "**A STUDY ON CONDUCTION ABNORMALITIES AND QRS DURATION IN PATIENTS WITH ISCHEMIC CARDIOMYOPATHY AND THEIR CORRELATION WITH EJECTION FRACTION**" submitted by **Dr. S.KARTHIKEYAN.,** to the Tamil Nadu Dr. MGR Medical University is in partial fulfillment of the requirement for the award of M.D. DEGREE (BRANCH -1) and is a bonafide research work carried out by him under direct supervision and guidance.

Signature of the Unit Chief
Dr. P.VIJAYARAGHAVAN. M.D.,

Signature of the Professor and HOD
Dr. S. MAGESHKUMAR. M.D.,

Signature of the Dean
Dr. S . GEETHALAKSHMI., M.D., Ph.D.,

DECLARATION

I solemnly declare that the dissertation entitled "**A STUDY ON CONDUCTION ABNORMALITIES AND QRS DURATION IN PATIENTS WITH ISCHEMIC CARDIOMYOPATHY AND THEIR CORRELATION WITH EJECTION FRACTION**" was done by me at the Government Stanley Medical College and Hospital during 2009-2011 under the guidance and supervision of **PROFESSOR Dr.P.VIJAYARAGHAVAN. M.D.** The dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of the requirements for the award of M.D.DEGREE (BRANCH – I) in general medicine.

Place : Chennai

Date : Dr. S.KARTHIKEYAN

ACKNOWLEDGEMENT

I owe my thanks to the Dean, Government Stanley Medical College and Hospital, Professor **Dr. S . GEETHALAKSHMI M.D., Ph.D.,** for allowing me to avail the facilities needed for my dissertation work.

I am extremely grateful to the Professor and Head of the Department of Internal medicine, Government Stanley Medical college and Hospital, **DR.S.MAGESHKUMAR M.D**., for permitting me to do the study and for being a constant source of encouragement .

My heartfelt gratitude to my unit chief **DR.P.VIJAYARAGHAVAN M.D**., Professor of Internal medicine, Government Stanley Medical College and Hospital for his wholesome support and valuable guidance without which I would not have completed this study .

I have a deep sense of gratitude towards Professor and Head of the Department of Cardiology Government Stanley Medical College and Hospital, **Dr. G. RAVISHANKAR M.D., DM.,** for approving this study and allowing me to work under his guidance .

I am extremely grateful to my unit assistant professors **Dr. K.THILAGAVATHY. MD., Dr. S. CHANDRASEKHAR MD.,** for their valuable suggestions and support.

Last but not the least I would like to sincerely thank all my fellow post graduate students for sharing their knowledge and my family without whose co operation this study would have been impossible to complete .

ABBREVIATIONS AND ACRONYMS

MI- Myocardial infarction

AWMI-Anterior wall myocardial infarction

IWMI-Inferior wall myocardial infarction

RVMI-Right ventricular myocardial infarction

ECG-Electrocardiogram

ECHO-Echocardiography

LBBB-Left bundle branch block

RBBB-Right bundle branch block

LAFB-Left anterior fascicular block

LPFB-Left posterior fascicular block

LVEF-Left ventricular ejection fraction

LVEDD-Left ventricular end diastolic diameter

RWMA-Regional wall motion abnormality

CONTENTS

INTRODUCTION

Electrocardiogram is an important tool cardiology. It not only helps to identify patients with ischemic cardiomyopathy but also prognosticate them. There are only a few studies which show an independent association of electrocardiogram findings with left ventricular systolic dysfunction. Studies in Indian population are lacking. Our study aims to observe electrocardiographic findings and then relate them with left ventricular systolic dysfunction in ischemic cardiomyopathy patients.

AIMS AND OBJECTIVES

1) To find out the prevalence of conduction abnormalities,

2) Measure QRS duration ,

in ischemic cardiomyopathy patients and correlate them with left ventricular ejection fraction(LVEF).

REVIEW OF LITERATURE

QRS COMPLEX

Depolarization or activation of the ventricles is reflected by the QRS complex in electrocardiogram.

THE NOMENCLATURE OF QRS DEFLECTIONS AND THEIR POTENTIAL FORMS

An initial downward deflection after P wave is termed as Q wave. An initial upward deflection after P wave is termed as R wave. The ensuing deflections are labeled in alphabetical sequence. The S wave is a negative deflection represents terminal part of ventricular activation. (1)

An initial small r wave followed by a large S wave is represented as rS complex. A complex with an approximately equal magnitude of R and S wave is termed as RS complex. A large R wave followed a small s wave is termed as Rs complex. A single wave complex which is totally positive is called as an R wave complex. A small initial downward deflection followed by a relatively tall upward deflection which in turn followed by a relatively large terminal deflection is termed as qRS complex. A complex with a relatively deep and wide initial negative deflection followed by a small terminal positivity is termed a Qr complex.

A complex with a complete negativity is termed as QS complex. A second positivity of QRS complex is termed as r'—r prime deflection. Thus an rS complex followed by a terminal positivity is termed as rSr' complex. When second terminal positivity is relatively tall then the complex is termed as rSR'. When the deflection is completely positive and notched it is termed as an RR'complex.(1)

GENESIS OF QRS COMPLEX

The activation of ventricles begins in the left sub endocardial region of lower third of the interventricular septum extending horizontally from left to right. It is opposed by a smaller activation force which occurs almost synchronously but a bit later and which arises in right sub endocardial region of the interventricular septum,spreading transversely through the septum from right to left.The much bigger left to right force dominates counteracting the smaller right to left force and resulting in effective vector which is directed transversely from left to right through the lower third of interventricular septum. This is called as septal force or septal vector.(1)

Activation of interventricular septum is then followed by activation of free walls of both ventricles.This occurs transversely from subendocardial to the subepicardial regions of the free walls of both

ventricles.This in simplified version is denoted by a relatively large force from right to left through the larger free wall of right ventricle which occurs synchronously with smaller opposing force which is directed from left to right. Through the smaller free wall of right ventricle.The larger right to left force of free left ventricular wall dominates and counteracts the smaller left to right force of right ventricular free wall.This results in a resultant or effective vector directed from right to left through free wall of left ventricle.Simplifying activation of ventricles is denoted by a small initial vector from left to right through the interventricular septum followed by a larger vector from right to left through the free wall of left ventricle.

THE EFFECT ON THE LEFT ORIENTED LEADS

The lead oriented to left ventricle say lead 1, AVL, V6 first senses relatively small resultant septal vector which is directed away from the positive pole of such a lead and consequently reflect a small downward deflection .This is followed by a large resultant vector of free left wall,which is directed towards the positive pole of such a lead.This will consequently result in large upward deflection, a relatively tall R wave.A left oriented lead will thus normally reflect a qR complex

THE EFFECT ON RIGHT ORIENTED LEADS

A lead oriented to right ventricle ,such as lead V1 orV2 will first sense the small resultant septal vector which is directed towards it,and will consequently reflect a small initial upward deflection, a small r wave .This is followed by a large resultant vector of free left wall , which is directed away from the right oriented lead and thus results in the relatively large downward deflection or S wave. A right oriented lead will therefore reflect normally an rS complex.

THE TRANSITION ZONE

The transition zone represents the electrocardiographic zone or lead which reflects the transition from rS pattern, usually recorded by a right oriented lead, the qR pattern usually recorded by a left oriented lead. It commonly occurs in or occasionally two of the mid precordial leads generally lead V3 and/ or lead V4. The transition pattern is usually an RS complex but sometimes it may be relatively bizarre.

QRS INTERVAL

The QRS interval is measured from start of q wave to the end of r wave. The upper limits for normal QRS complex is 0.10s. In general the QRS complex is short in childhood and longer in senescence.(2)

ANATOMY OF SPECIALIZED CARDIAC CONDUCTION SYSTEM

SINO ATRIAL NODE

The sino atrial node (S.A node) is the primary pacemaker of the heart.In adult it is approximately 10 – 20 mm long and elliptical in shape.One end lies just beneath the epicardium and opposite lying just beneath the endocardial surface of the superior and right side of the atrium near the root of superior vena cava and the base of right atrial appendage.

Microscopically it consists of network of dense collagen material containing Purkinje like fiber and special group of primary pacemaker cells. In adults the usual resting discharge rate is around 60—100.

INTER NODAL TRACTS

There are three well described inter nodal pathways namely

a) Anterior
b) Middle
c) Posterior

Experimental work suggest that anterior and middle are the most important in conducting impulse from S.A node to the A.V node(3)

The anterior internodal tract leaves the sinus node in an anterior position and curves around the superior vena cava and anterior wall of right atrium, where it meets the Bachman bundle, the major inter atrial bundle.These fibers then divide into two branches one of which goes into left atrium as Bachman bundle and the other continues down as the inter atrial septum and enters the anterior portion of A V node.

The middle inter nodal tract, otherwise called as the Wenkebachs bundle, leaves the dorsal and posterior margins of sinus node and curves posterior to SVC extending down the dorsal portion of inter atrial septum,giving off a few sparse fibers to left atrium and then to the superior margin of the AV node

The posterior inter nodal tract leaves the posterior margin of sinus node enters the crista terminalis and the region of Eustachian ridge and eventually reaches the AV node in its lower posterior portion.

All the three of intermodal tracts have fibers that meet at the atrio nodal junction. Some investigators believe that at this junction and at the distal AV node—His bundle junction the greatest delay in the transmission of impulse occurs and that this delay is not in the AV node proper.

In junctional region there are also a great number of P cells.In abnormal situations these cells might take up the role of S.A node as

pacemaker and give rise to rhythms called as junctional rhythms. These rhythms were called as the AV nodal rhythms although they arise from the junctional areas and not the AV node proper.

AV node

The AV node is smaller in size than the S.A node, measuring approximately 3mm by 6mm.Itis an elliptical structure located in the endocardial surface of the right side of the interatrial septum just anterior to the ostium of coronary sinus and directly above the insertion of the septal leaflet of tricuspid valve. Microscopically it is made up of interlacing Purkinje like fibers with much less collagen than in sinus node. Hoffman and Cranefield divided the AV node into three electrophysiological parts according to action potentials and the response to chemical and electrical stimulation (4)

a) upper junctional area --AN region
b) the N region
c) the lower junctional area -- NH region

COMMON BUNDLE OF HIS

The bundle of HIS is an extension of the tail of the AV node. It is approximately 2 cm long in adult, but its size varies considerably. It is located in the endocardial surface of the right side of the inter atrial septum

and extends downwards and leftwards to enter the interventricular septum near the junction of the muscular and fibrous portions of the ventricular septum.The parallel fibers of HIS bundle are separated by fine collagen septa (5)

The bundle of HIS divides into right and left branches. The right bundle extends down the right side of the ventricular septum as a single stalk covers by connective tissue, until it reaches the anterior papillary muscle at the apex of right ventricle. There it divides into small arborizations of the Purkinje system that spread over the endocardial surface of the ventricular wall and right ventricular surface of ventricular septum.The upper and distal third lie close to the endocardium whereas the middle third lies deep inside the myocardium.As a result of such proximity to the endocardium in the majority of its portions any slight rise in intra cavitary pressures may alter conduction and produce a conduction block.Similarly mechanical factors such as stretching occurring in heart failure can result in conduction abnormalities of right bundle branch.

The left bundle branch in contrast to right branches almost immediately after bifurcation. The main trunk of left bundle is narrow at its origin emerging in the sub aortic region at the left of commissure between the right coronary cusp and non coronary cusp. It widens prior to branching into anterior division and posterior division.The anterior

division is thin and long lying below the aortic valve in left ventricular outflow tract. The posterior division is much thicker and shorter lying mostly in left ventricular inflow tract. Most adults have a septal fascicle although this is much variable.In general fibers of posterior division separate proximally high in branching part of his bundle(6)

The actual bundle bifurcation represents division of distal common bundle into right bundle branch and posterior and anterior divisions of left bundle branch.Each division terminates in respective papillary muscle of mitral valve.The divisions are now termed fascicles.Each major fascicle is responsible for activation of a portion of septum. Thus intraventricular conduction system consists of bundle of his the right bundle and left bundle.The left bundle has an anterior fascicle, posterior fascicle and occasionally septal fascicle. The trifascicular conduction system refers to three main fascicles,the right bundle branch and two major fascicles of left bundle branch.

Because of such conduction system, activation of ventricular musculature occurs initially at the middle third of left ventricular side of interventricular septum and subsequently the free walls of left ventricle. (7)

BUNDLE OF KENT

Right and left accessory pathways bundles of Kent which directly connects the right and left atria to respective ventricles, have also been identified.Proof that Kent bundles probably conduct impulses from atria to ventricles directly (pre excitation) has been obtained by mapping epicardial excitation of the exposed heart at the time of surgery and by intra cardiac electrograms.

MAHAIM FIBERS

Other anomalous pathways connecting atria and ventricles are the specific paraseptal fibers of MAHAIM, providing direct connections between the lower AV node, the his bundle and the bundle branches within the ventricular septum.

JAMES TRACT

These pathways connect the atria with lower AV node or bundle of his.

ARTERIAL BLOOD SUPPLY OF THE SPECIALISED CONDUCTION SYSTEM

The arterial blood supply to the specialized conduction system is critical to its proper function. Disorders of blood supply produced by

coronary artery disease disturb the function of conduction and give rise to arrhythmias. The artery to sinus node arises from right coronary artery in 54% to 60% of cases, and from left coronary artery in the remainder. The artery to sinus node runs along the anterior portion of interatrial septum, circles the base of the superior vena cava, and then divides into multiple nutrient branches that supply the area of the sinus node.As previously mentioned, the most interesting feature of sinus node is its constant relationship to its large centrally located artery.

The AV node artery arises from right coronary artery in about 86% of persons,from the left coronary artery in 12% and from both arteries in 2%.The AV nodal artery usually leaves the coronary artery at right angle at the crux of heart (the U turn) near the junction of inter atrial and inter ventricular septum posterior to coronary sinus.The right angle origin of this artery creates a point of mechanical stress and pre disposes this artery to atheromatous changes at this point.The incidence of AV conduction disturbances in inferior myocardial infarction because such infarctions are secondary to occlusion right coronary artery,the most common supply to AV node.(8)

The left anterior descending artery and its septal perforating branches compose the primary circulation to bundle of His, the right bundle and the left bundle. When right coronary artery gives rise to

posterior coronary artery, its septal perforators supply the posterior fascicle of the left bundle as well.Therefore the posterior fascicle can be supplied by right as well as left coronary artery as well.When the circumflex gives rise to posterior descending artery, the posterior fascicle is supplied by both major branches of left coronary artery. The anterior descending coronary artery supplies the Purkinje system in the septum.The incidence serious AV blocks and intraventricular conduction defects is higher in anterior myocardial infarctions since these infarctions are due to occlusions of branches of anterior descending coronary artery.

RIGHT BUNDLE BRANCH BLOCK

Right bundle branch block is a delay or block in conduction within the right bundle branch.

- A delay of conduction manifests as incomplete right bundle branch block
- A block or compete interruption of conduction manifests as complete right bundle branch block.

The mode of intraventricular conduction with these two forms of right bundle branch block differs considerably.

There are two theories about sequence of activation in right bundle bundle branch block

- The conduction delay occurs in the septum due to muscle cell to muscle cell conduction through the septum and right ventricular free wall
- The second theory postulates that there is a leak at the site of bundle branch block and as a consequence right ventricular activation starts at the site of block and progresses peripherally.

Grant & Dodge and other proponents of the second theory postulate that the primary block is located peripheral to the main right bundle branch block and that excitation process on reaching the site of block leaks into the surrounding regions of the right ventricle and spreads by abnormal routes and at a slower fiber to fiber trans myocardial process(9)

COMPLETE RIGHT BUNDLE BRANCH BLOCK

Complete right bundle branch is characterized by the following

1. Lead V1 reflects a tall wide and frequently notched R' deflection
2. The left oriented leads, V5 and V6 as well as standard lead1 reflect a prominent delayed and widened S wave.
3. The QRS duration is increased to 0.14 sec or longer.

The mechanism of ventricular activation

With complete right bundle block ,as with normal intraventricular conduction , activation of the ventricles begin in left lower third of interventricular septum and spreads transversely from left to right through the septum.This left to right vector however is now no longer opposed by the normal smaller right to left septal vector which originates from the right bundle branch and which arises in the right side of ventricular septum.And since the left to right septal vector is no longer opposed it is therefore minimally increased in magnitude.This may result in a slightly more prominent initial r wave in lead v2 which is the lead that is most aligned with the vector.

Normal left paraseptal activation occurs next, spreading transversely from endocardial to epicardial surfaces. This is followed by

endocardial to epicardial activation of free wall of left ventricle.This free wall left ventricular vector is directed to left.

Activation of right side of interventricular septum and the right and free wall is effected by activation front which arises in the left side of interventricular septum. This crosses, literally jumps a physiological intraseptal barrier and is then conducted through ordinary myocardial tissue and not specialized conduction tissue, within the right ventricular wall. (11)

This right sided activation is consequently slow and anomalous in character, tending to be longitudinal or tangential rather than transverse (endocardial – epicardial). The Purkinje system is programmed for rapid transmission of activation process from the central distributing point which is in the sub endocardium.Activation entering the system from other direction i.e.within myocardium, is also transmitted in a bizarre, relatively slow manner and also is ineffective (12).

a) This results in slow abnormal depolarization of right side of a) interventricular septum,
b) The right paraseptal region
c) The free wall of right ventricle.

This slow abnormal depolarization is reflected electrocardiographically by large slow inscribed vectors whichare directed anteriorly and to the right. The abnormal right paraseptal vector occurs simultaneously with , but is opposite in direction to the vector of left free wall. These vectors thus oppose and tend to neutralize each other. This results in marked diminution of S wave in lead V1, which in advanced degree of right bundle branch block may disappear completely. This process also results in attenuation of R wave in V6.

Unlike the effect on lead V1, the R wave in lead V6 is diminished but not abolished completely. This is because the lead V6 is oriented to left free wall of left ventricle and is thus influenced by degree of paraseptal vector which also contributes to the positivity of R wave V6. Right paraseptal activation is followed by abnormal and slow activation of right free wall. These results in large unopposed vector directed anteriorly and to right. It will be reflected by a wide R' deflection in lead V1 and a prominent slurred and delayed S wave in lead V6.This slow anomalous form of activation is also responsible for the marked notchings and slurrings of terminal bizarre and widened deflections.

Leads oriented to right ventricle, for example leadV1 reflect the following

1. There is an initial small r wave due to left septal depolarization
2. This is followed by an S wave or more likely an s wave which is due to mainly to depolarization of left free wall. This S wave is attenuated and may even disappear completely.
3. This is a terminal bizarre and slurred R' wave – due to late and anomalous right septal and right free wall depolarization.

Leads oriented to left ventricle V5, V6 and standard lead 1, reflect the following

1. There is a small initial q wave, due left septal depolarization
2. There is a relatively tall R wave,mainly due to depolarization of left free wall.
3. There is a terminal bizarre and slurred S wave due to late anomalous right septal and free wall activation

THE QRS DURATION

The minimum QRS duration in case of complete right bundle branch block is usually stated to be 0.12 s. This criterion however requires reappraisal. For the diagnosis of complete right bundle branch block the R'deflection must also be wide and plateau shaped or notched. Since the duration of R' deflection in case of incomplete right bundle branch block may well be 0.04 sec, the duration of R' deflection is therefore expected to

be about 0.06 sec. And, since the basic initial vectors, excluding the terminal vector due exclusively to the block are usually 0.08 sec in duration, the total duration of QRS complex in complete right bundle branch block could be expected to be 0.14 sec or more. This also requires further evaluation. (1)

VARIANTS

The following variants of bundle branch block have been

1. The Wilson – type block

This is characterized by relatively tall and narrow initial QRS complex, followed by wide terminal S wave in standard lead1 .This merely reflects the manifestation where initial QRS forcesare directed to the region of 0 degree on hexaxial reference system.

2. Right bundle branch block with left posterior hemiblock
3. In this form of right bundle branch block also known as uncommon form of right bundle branch block, the terminal QRS forces are of much greater magnitude than in conventional form. This is further associated with right axis deviation of initial QRS forces. The manifestation is in effect is a right bundle branch block with a left posterior hemiblock.

INCOMPLETE RIGHT BUNDLE BRANCH BLOCK

In the case of incomplete right bundle branch block, conduction through right bundle branch and its ramifications is still possible but is delayed. This contrasts with complete right bundle branch block, where conduction through right specialized system is no longer possible but occurs through ordinary myocardial tissue.

The type of electrocardiographic manifestation which occurs in incomplete right bundle branch block will depend upon the degree of delay within the right bundle branch. Progressively increasing delay of conduction within the right bundle branch will result in a progressive sequence of electrocardiographic manifestations. They are considered below with reference to right oriented leads – lead V1 and V2, especially lead V2.

THE ELECTROCARDIOGRAPHIC MANIFESTATIONS OF INCOMPLETE RIGHT BUNDLE BRANCH BLOCK

With progressive increase in delay within right bundle branch, the following sequence of progressively increasing incomplete right bundle branch block manifests

1. There is diminution of the S wave in leadV2. This the earliest sign of incomplete right bundle branch block. (13,14). This may be the

only sign of early incomplete right bundle branch block(15). This is usually evident when incomplete right bundle branch block manifests as an expression of aberrant ventricular conduction. It is however very difficult to judge the diminution of S wave in lead V2 when there is aberrant ventricular conduction. Serial tracings then become necessary. And even under these circumstances one cannot be absolutely be certain of diminution of S wave, since the effect of a slightly different electrode placement may also play a very important role in diminution of S wave sometimes.

2. The diminution of S wave is followed by slurring of upstroke of S wave in lead V2. This is further associated with diminution of S wave.
3. A small r' deflection appears in lead V2. This will result in rsr' configuration. This diminutive r' is not widened and is usually associated with further attenuation of S wave
4. The amplitude of R' deflection in lead V2 then increases. This is a more marked manifestation. The S wave is further diminished and the configuration is further that of rsR' complex. While QRS complex reflects slight increase in duration it is not increased beyond 0.11 sec. the R' deflection itself is not widened beyond 0.04

sec. While R' deflection becomes widened and notched eventually the right bundle branch may be considered complete

The development of right bundle branch block is thus characterized empirically by two major manifestations in lead V2:

1. Progressive diminution of S wave.
2. Progressive enlargement of r' or R' with final widening of this deflection. The embryonic r first appears as a slur on the upstroke of S wave. This process slightly later than and overlaps with the process leading to diminution of S wave.

MECHANISM

In case of incomplete right bundle branch block conduction is still possible but delayed. A small delay in right bundle branch causes a delay in inscription of right paraseptal vector. As a result this vector occurs synchronously with free wall forces. A slight delay in conduction through right bundle branch will thus cause a diminution of S wave in lead2.

There should also be a diminution of R wave in leadV6. While this can occur the manifestation is not consistent. A possible reason for this lack of consistency is that lead V6 is oriented more obliquely, i.e. towards the left paraseptal vector as well as left wall vector. It will therefore influenced by the left paraseptal vector, which will contribute to the amplitude of R wave

in this lead. Further more lead V6 is more remote from the heart than V2 and is thus subject to more variations in amplitude due to

1. Body build
2. The amount of intervening lung tissue

The same probably applies to V1 but requires further study. Since the right paraseptal vector is the most affected, the effect will be most likely evident in lead V2, which is directly oriented to this vector. As the delay within the right bundle branch increases,activation of free wall of right ventricle becomes increasingly delayed. And, when the delay is of such degree that activation of free wall of the right ventricle occurs after activation of left ventricle free wall an r' deflection appears in right oriented leads. This is because right free wall activation is no longer opposed completely by left free wall activation. The R' deflection becomes increasingly taller since larger part of right free wall activation will occur after left free wall. The R' deflection of maximal delay, however is not widened beyond 0.04 sec, and if it is so the block is regarded as a complete block.

THE SIGNIFICANCE OF ANATOMICAL LENGTH OF RIGHT BUNDLE BRANCH

It is becoming increasingly clear that slowing of conduction is not the only cause of conduction delay in the genesis of incomplete right bundle branch block. The length of right bundle branch also plays a significant role. The normal right bundle is about 40 mm in length. The normal conduction velocity in right bundle branch is 2 meters per second. Thus for each centimeter increase in length of right bundle, the conduction time through the bundle will increase by 0.005 sec. if, for example,the length of right bundle is increased by 4 cm – not an uncommon occurrence with chronic right ventricular dilatation – the conduction time within the right bundle branch will increase by 0.02 sec. Therefore to begin with, uncomplicated intraventricular conduction results in a QRS duration of 0.09 sec, a 4 cm increase in length of right bundle branch alone will increase the QRS duration by 0.11 sec.

Thus the anatomical factor is clearly significant when there is dilatation of ventricle due to volume or systolic overload as in:

1) Atrial septal defect
2) Cor-pulmonale
3) Tricuspid insufficiency

THE SIGNIFICANCE OF RIGHT BUNDLE BRANCH BLOCK

Right bundle branch may occur in the absence of heart disease. It has been reported in normal air force personnel with an incidence of 1.5/1000 between the ages of 20 to 40 years, and 2.9/1000 in individuals over 40 years (19). However it may be associated with

1) Coronary artery disease
2) Various cardiomyopathies
3) Ebstein's anomaly
4) Atrial septal defect
5) Persistent atrioventricularis communis
6) Acute pulmonary embolism

Consequently, right bundle branch must always be evaluated in the perspective of the associated clinical and electrocardiographic manifestations.

LEFT BUNDLE BRANCH BLOCK

Left bundle branch block is the result of delay or interruption of conduction within the left bundle branch.

1) A delay of conduction manifests as incomplete bundle branch block

2) A complete interruption or block of conduction results in complete bundle branch block.

COMPLETE LEFT BUNDLE BRANCH BLOCK

With complete left bundle branch block, activation of left side of interventricular septum and free left wall is delayed and anomalous in character. It has been shown that the Purkinje system is in fact used in fact used in anomalous form of left bundle branch activation. The activation process tends to be longitudinal or tangential or oblique rather than centrifugal. It would seem that the Purkinje system is programmed for transmission of activation process from a central distributing point, which in case of left ventricle is normally the left ventricular sub-endocardium. Activation entering the system from another direction, i.e. from within the myocardium , is also transmitted but not in a very effective manner. Thus there are three components of ventricular activation in complete left bundle branch block. These in chronological sequence are:

1) Right septal activation
2) Delayed and anomalous left septal activation
3) Delayed and anomalous activation of left ventricular free wall

THE ELECTROCARDIOGRAPHIC MANIFESTATIONS

The QRS duration

The QRS duration is prolonged to 0.12 sec or more and may be as long as 0.20 sec. this is due to delayed and anomalous activation of

a) Left septal mass
b) The free wall of left ventricle

The QRS expression in various leads

Left oriented leads V5 and V6 as well as standard lead1 theoretically reflect three positive deflections,

a) r1,
b) R2,
c) R3

The first deflection is rarely seen.

Lead aVL may reflect the following

1) An RsR complex. The represents a notched plateau. This may at times be preceded bya small initial q wave. This represents additional manifestation of first component of leftbundle branch block activation or more likely the vector of right paraseptal mass.

2) A wide but unnotched complex

Leads V1 and V2 usually reflect a widened notched QS complex or an rS complex. The small initial R wave is more prominent in lead V2

Leads which are oriented to right paraseptal region notably lead V2 may reflect the rather prominent right paraseptal vector as a conspicuous initial r wave.

The secondary ST segment T wave changes

The ST segment and T wave are opposite in direction to the terminal QRS deflection. These are secondary phenomenon, i.e. they occur secondarily to abnormal intraventricular conduction and do not indicate primary ST segment or T wave abnormality. Thus in leads oriented to left ventricle (leads V5 and V6) the ST segment is depressed and is often convex upwards minimally. The T wave is inverted with a blunt nadir. The T wave is asymmetrical in leads oriented to right ventricle, and ST is elevated and often minimally concave upward. The T wave is upright asymmetrical and relatively with a blunt apex.

INCOMPLETE LEFT BUNDLE BRANCH BLOCK

In case in complete left bundle branch block, conduction through the left bundle branch and its ramifications is still possible but is delayed. This

is in contrast to complete left bundle branch block where conduction through the left sided specialized conducting system is no longer possible but occurs through ordinary myocardial tissue. The type of electrocardiographic manifestation that occurs with incomplete bundle branch block will depend upon the degree of delay within the left bundle branch. Progressively increase in delay of conduction within the left bundle branch will result in a progressive sequence of electrocardiographic manifestations (20).

The earliest manifestations of incomplete bundle branch block

The first manifestations of incomplete left bundle branch block are the following

1) The small initial q wave of the qR complex in the left oriented leads V5 & V6and standard lead 1 disappears. This will result in single tall R wave
2) The small r wave of the initial rS complex in lead V1 disappears resulting in QS complex.

   These manifestations are due to a delay in the formation and inscription of normal left sided septal vector. This means that the normal right sided septal vector now has more time to develop and it equals the magnitude of left sided vector. Thus the two septal

vectors thus cancel or nullify each other, there by resulting in disappearance of effective or resultant normal left to right septal vector.

The manifestations of more advanced form of incomplete left bundle branch block

With further progression of the incomplete left bundle branch block a slur appears on the upstroke of QRS complex. This slur becomes increasingly prominent and is accompanied by widening and eventual notching of QRS complex until fully developed manifestation of complete left bundle branch block is obtained.

Such manifestations are due to the increasing dominance of right septal vector, which penetrates intraseptal electrophysiological barrier to an increasing degree. It is permitted to play a more dominant role due to increasing delay within the left bundle branch.

LEFT BUNDLE BRANCH BLOCK WITH LEFT ATRIAL ENLARGEMENT

The electrocardiographic manifestation of left atrial enlargement in the presence of left bundle branch block is a useful pointer to the presence of left ventricular hypertrophy, which is most likely due to systemic hypertension (21). Left atrial is also a pointer to more severe

cardiovascular disease, such as would be reflected by cardiomegaly and congestive heart failure.

SIGNIFICANCE OF COMPLETE LEFT BUNDLE BRANCH BLOCK

Complete left bundle branch block indicates organic heart disease. It is commonly associated with,

a) Ischemic heart disease

b) Hypertensive heart disease

ARBORIZATION BLOCK

When bundle branch block is associated with low amplitude complexes, the condition is sometimes referred as arborization block. This form of bundle branch block has been incorrectly attributed to block of Purkinje fibers. The term is rarely used today.

THE HEMIBLOCKS (FASCICULAR BLOCKS)

The hemiblocks or fascicular block is a delay in conduction within one of the major divisions or fasciculi of left bundle branch. It therefore constitutes a form intraventricular conduction defect.

Left anterior hemiblock

When conduction is delayed or interrupted in anterosuperior division of left bundle branch it is termed as left anterior hemiblock. The interruption may be due to fibrosis, for example

1) Coronary artery disease
2) Cardiomyopathy
3) Hypertension
4) Congestive cardiac failure

It also may be due to myocardial infarction, where it is called as peri-infarction block. When such interruption or delay occurs activation will proceed predominantly through the posterosuperior division of left bundle branch, resulting in mean frontal QRS axis that is directed superiorly and to the left— a left axis deviation.

The basic effects

The first part of the ventricle to be activated with left anterior hemiblock is inferior septal mass and the inferior region of left ventricular free wall, i.e. the posteroinferior region of left ventricle. This results in initial QRS forces which directed inferiorly and, commonly to right

The initial activation is followed by delayed activation of free left ventricular free wall: a regional delay in the depolarization of left anterosuperior wall. This results in dominant and terminal QRS forces being directed:

1) Superiorly and to the left in the frontal plane—left axis deviation
2) Somewhat posteriorly in the horizontal plane

The basic electrocardiographic characteristics

Left anterior hemiblock results in the following basic disorders of intra ventricular conduction,

1) Left axis deviation of mean frontal plane QRS axis.
2) Delay in the activation of free left ventricular wall, particularly in the high anterolateral region resulting in increased ventricular activation time.
3) An increase in magnitude and change in direction of the initial 0.02 sec vector, which is directed inferiorly and commonly somewhat to the rightwards
4) Secondary T wave changes.

The effects on the QRS complex

Left anterior hemiblock results in the following modifications of QRS complex,

1) Left QRS deviation
2) A prominent initial QRS vector which is directed inferiorly and commonly to the right.
3) A delay in the inscription of intrinsicoid deflection.
4) Slight slurring or irregularity of the QRS limbs
5) A planar re orientation of QRS forces
6) Increased magnitude of dominant QRS deflection
7) A slight increase in QRS duration

Left QRS axis deviation

The mean frontal plane QRS axis is deviated superiorly and to the left. The mean QRS may be located within arrange extending from -30 counter clockwise to -80 degree. The left QRS axis deviation will be reflected empirically by:

1) Deep terminal S waves in lead 2, 3 and aVF
2) Tall R wave in lead aVL

Since the QRS is more aligned with standard lead 3 lead axis, the S wave is of greater amplitude – deeper in lead 3 than in lead 2. When S wave is deeper in lead 2 than lead 3, it connotes a mean frontal QRS axis which is directed superiorly and to the right -- to the northwest region. When this occurs pure uncomplicated left anterior hemiblock is unlikely.

It has been stated that between 0 to -30 degree can manifest in following people,

1) Stockily built people
2) Ascites
3) Pregnancy
4) Obesity.

This is because abdominal distension will displace the heart upwards resulting in so called a horizontal heart and manifest with minor degree of left axis deviation. However this has not been substantiated. In a study conducted in pregnant women at full term with maximal abdominal distension revealed no such axis deviation.It is clear that left anterior hemiblock occurs as a manifestations of overt heart disease the mean frontal QRS axis is directed to -30 degree. The other causes of left axis deviation are,

1) Inferior wall myocardial infarction
2) Wolf Parkinson White syndrome
3) Pacing rom apex of right ventricle
4) Pacing from apex of left ventricle
5) Ectopic rhythm from apex of left ventricle
6) COPD
7) Congenital heart diseases which includes,
    a) Endocardial cushion defects
    b) Persistent atrioventricularis communis
    c) Hypoplastic right ventricle
    d) Single ventricle
    e) Corrected transposition of great vessels

Comments

The commonest of left axis deviation is left anterior hemiblock. This is mostly due to fibrosis which interrupts the anterosuperior division of left bundle branch. This may be due to,

1) Chronic coronary insufficiency
2) Chronic cardiac failure
3) Long standing systemic hypertension
4) Chronic cardiomyopathy
5) Subclinical coronary artery disease in elderly.

Prominent initial QRS vector directed inferiorly and commonly to the right in the frontal plane

The initial 0.02 sec of QRS vector is directed inferiorly and commonly to the right to the region of +90 to +120 degree on the frontal plane hexaxial system. This will result in the following,

1) Prominent initial Q waves appear in standard lead1and especially lead aVL. In other words the normal small q waves in these lead becomes accentuated.
2) Prominent initial r waves appear in standard leads 2, lead 3, and lead aVF in hexaxial system.

The prominent initial QRS vector may be obliterated by,

1) Complete left bundle branch block.
2) Inferior wall myocardial infarction
3) Superiorly directed delta wave of Wolf Parkinson White syndrome.

The delay in the inscription of intrinsicoid deflection

There is a delay in the inscription of intrinsicoid deflection---delayed R peak time—in leads oriented to high lateral or superior aspect of left ventricle particularly lead aVL. The intrinsicoid deflection may be delayed 0.045 sec or longer. The delay may also be reflected in lead aVR

due to incorporation of terminal R wave, and may at times be seen only in lead aVR.

Slight slurring or irregularity of QRS limbs

There may be slurring, notchings and irregularities in lead aVR and lead 1 as lead V5, lead V6. There may be slurred terminal r or r' deflection in lead aVR.

Slight increase in QRS duration

There may be slight increase in QRS duration but is usually less than 0.11 sec. The criterion for diagnosis of uncomplicated left anterior hemiblock requires a QRS duration of less 0.11 sec since complicating factors such as,

1) Myocardial fibrosis of left free wall
2) Right ventricular hypertrophy
3) Right bundle branch

may further prolong QRS duration.

The effect on left precordial leads V5 ,V6

Left anterior hemiblock results in the following effects in precordial leads,

1) The tendency for normal q waves which appear normally in V5,V6 to disappear.
2) Attenuation of R waves in left oriented precordial leads, a reflection superiorly directed QRS vector.
3) The development of prominent S waves in left precordial leads, a reflection superiorly directed QRS vector.
4) There is also slurring of S wave in V5, V6.

The significance of left anterior hemiblock with increased QRS amplitude

High grades of left anterior hemiblock will merely increase the duration end amplitude of QRS deflections. Thus when S wave increases in amplitude in lead 3 greater than 1.5mm the manifestation may be due to,

1) Advanced left anterior hemiblock
2) Associated left ventricular hypertrophy.

Peri – infarction block

It refers to delay in conduction within the tissue surrounding the infarcted region.

Types of peri-infarction block

1. Focal block

There is local disturbance of conduction around infarction.

2. Divisional block

The infarction interrupts anterosuperior division of left bundle branch. It occurs with

a) Anterolateral myocardial infarction
b) Inferior wall myocardial infarction

LEFT POSTERIOR HEMIBLOCK

Here the conduction is interrupted in the posteroinferior division of left bundle branch. The basic effects are

a. Right axis deviation of mean QRS vector.
b. Left superior deviation of initial QRS vector
c. Secondary repolarization changes of T wave .

METHODOLOGY

Individuals admitted in medical wards and cardiology ward satisfying the inclusion criteria below from April 2011 to September 2011 are studied. ECG is taken and ejection fraction is measured echocardiography in them. The results are analyzed statistically.

INCLUSION CRITERIA

1) History of myocardial infarction in the past which is defined as,

   Typical rise and/or fall of biochemical markers of myocardial necrosis with at least one of the following

   a) Ischemic symptoms

   b) Development of pathological Q waves in ECG

   c) ECG changes indicative of ischemia (ST segment elevation or depression)

   d) Echocardiogram evidence of loss of viable myocardium

2) Echocardiogram showing,

   a) Left ventricular ejection fraction less than 40%

   b) Presence of regional wall motion abnormality(RWMA)

EXCLUSION CRITERIA

1) Rheumatic heart disease – mitral regurgitation/aortic regurgitation
2) Thyroid disease
3) Chronic alcohol abuse >3 years
4) Documented recurrent episodes of ventricular tachycardia/supraventricular tachycardia
5) Women in postpartum period

STATISTICAL ANALYSIS

All the results are presented as mean ± SD or percentage of the total. Data are tabulated using excel. Statistical analysis was done using SPSS software version 20. The difference between categorical variables was tested for significance by Fisher exact test. Continuous variables were compared using ANOVA test. A p value of < 0.005 is considered significant. Correlation was done based on Pearson correlation and Spearman correlation.

RESULTS AND ANALYSIS

TABLE: 1

AGE GROUPS	SEX			
	Female		Male	
	n	Percentage	n	Percentage
<50	2	4.0%	2	4.0%
51-60	9	18.0%	12	24.0%
61-70	10	20.0%	7	14.0%
>70	5	10.0%	3	6.0%

This table shows

1) The percentage of study population in different age groups

2) The percentage of study population in both sexes

TABLE:2

Clinical variable		MI-TYPE					
		AWMI		IWMI		IWMI/RVMI	
		n	N %	n	N %	n	N %
Sex	Female	16	32.0%	7	14.0%	3	6.0%
	Male	16	32.0%	6	12.0%	2	4.0%
Dm	No	17	34.0%	8	16.0%	1	2.0%
	Yes	15	30.0%	5	10.0%	4	8.0%
Ht	No	13	26.0%	6	12.0%	5	10.0%
	Yes	19	38.0%	7	14.0%	0	-
Smoking	No	19	38.0%	7	14.0%	4	8.0%
	Yes	13	26.0%	6	12.0%	1	2.0%
Alcohol	No	20	40.0%	6	12.0%	4	8.0%
	Yes	12	24.0%	7	14.0%	1	2.0%
Conduction abnormality	LBBB	26	52.0%	13	26.0%	0	-
	LBBB+ LAFB	6	12.0%	0	-	0	-
	RBBB	0	-	0	-	3	6.0%
	RBBB+ LAFB	0	-	0	-	2	4.0%

This table shows, the association of different variables with type of myocardial infarctions.

Table :3

Clinical variable		QRS DURATION (ms)				
		Mean	**Median**	**Standard Deviation**	**Range**	**Fisher exact test p**
Sex	Female	153	160	14	40	0.408
	Male	160	160	18	60	
Diabetes	No	158	160	17	60	0.294
	Yes	154	160	15	40	
Height	No	154	160	14	40	0.735
	Yes	158	160	18	60	
Smoking	No	153	160	13	40	0.300
	Yes	161	160	19	60	
Alcohol	No	155	160	14	40	0.457
	Yes	159	160	19	60	
Conduction abnormality	LBBB	153	160	12	40	<0.001
	LBBB+ LAFB	187	180	10	20	
	RBBB	140	140	0	0	
	RBBB+ LAFB	150	150	14	20	

This table shows the association of different variables with QRS duration.

Table :4

AGE GROUPS	MI-TYPE					
	AWMI		IWMI		IWMI/RVMI	
	Count	Table N %	Count	Table N %	Count	Table N %
<50	2	4.0%	1	2.0%	1	2.0%
51-60	14	28.0%	5	10.0%	2	4.0%
61-70	12	24.0%	5	10.0%	0	0.0%
>70	4	8.0%	2	4.0%	2	4.0%

This table shows the percentage of different type of myocardial infarctions in various age groups.

Table :5

AGE GROUPS	DURATION FOLLOWING MI	
	Mean	**Standard Deviation**
<50	5	2
51-60	5	1
61-70	5	1
>70	5	1

This table shows, the mean duration following myocardial infarction in different age groups of study population.

Table :6

AGE GROUPS			QRS DURATION (ms)		
			Mean	**Standard Deviation**	**Median**
<50	SEX	F	140	0	140
		M	150	14	150
51-60	SEX	F	158	19	160
		M	163	14	160
61-70	SEX	F	154	10	160
		M	160	23	160
>70	SEX	F	148	11	140
		M	153	23	140

This table shows, the mean and median QRS duration in different age groups of study population.

Table :7

AGE GROUPS			EJECTION FRACTION %	
			Mean	**Standard Deviation**
<50	SEX	F	32	8
		M	36	3
51-60	SEX	F	32	5
		M	31	6
61-70	SEX	F	33	3
		M	33	5
>70	SEX	F	35	2
		M	33	10

This table shows the, the mean ejection fraction in different age groups of the study population.

Table :8

AGE GROUPS			LVEDD cm	
			Mean	**Standard Deviation**
<50	SEX	F	5.7	.4
		M	6.1	.1
51-60	SEX	F	5.9	.3
		M	6.4	.3
61-70	SEX	F	5.7	.3
		M	6.4	.3
>70	SEX	F	5.7	.3
		M	6.3	.5

This table shows the, the mean LVEDD in different age groups of study population.

Table :9

MI-TYPE	QRS DURATION ms		
	Mean	**Standard Deviation**	**Median**
AWMI	160	17	160
IWMI	152	13	160
IWMI/RVMI	144	9	140
			P=0.490

This table shows the mean and median QRS duration in various types if myocardial infarction.

Table :10

QRS DURATION (ms)	AGE	
	Mean	**Standard Deviation**
140	63	9
160	62	8
180	60	8
200	61	8
	F=0.109	p=0.954

This table shows the relationship of QRS duration with age. This relationship is not statistically significant, p=0.954

Table :11

QRS DURATION (ms)	DURATION FOLLOWING MI	
	Mean	**Standard Deviation**
140	5	1
160	5	1
180	6	0
200	7	1
	F=2.847	**p=0.048**

This table shows the relationship between QRS duration and duration following myocardial infarction. The relationship is statistically significant, p=0.048.

Table :12

QRS DURATION ms	EJECTION FRACTION %	
	Mean	**Standard Deviation**
140	35	5
160	33	3
180	27	5
200	23	7
	F=9.252	p<0.001

This table shows the relationship between QRS duration and ejection fraction.The relationship is statistically significant, p<0.001.

Table :13

QRS DURATION ms	LVEDD cm	
	Mean	**Standard Deviation**
140	5.8	.3
160	6.1	.3
180	6.3	.5
200	6.8	.4
	F=5.688	p<0.001

This table shows the relationship between QRS duration and LVEDD.The relationship is statistically significant, p<0.001.

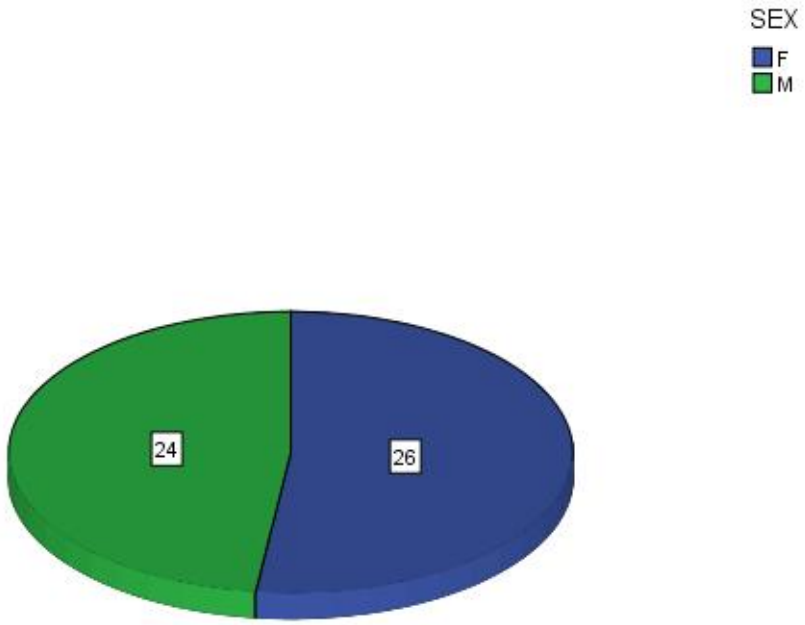
Table :14

QRS duration	Pearson's correlation coefficient	P value	Spearman's correlation coefficient	P value
Age	-0.076	0.598	-0.064	0.657
Duration following MI	0.394	0.005	0.443	0.001
LVEF	-0.594	<0.001	-0.532	<0.001
LVEDD	0.537	<0.001	0.439	0.001

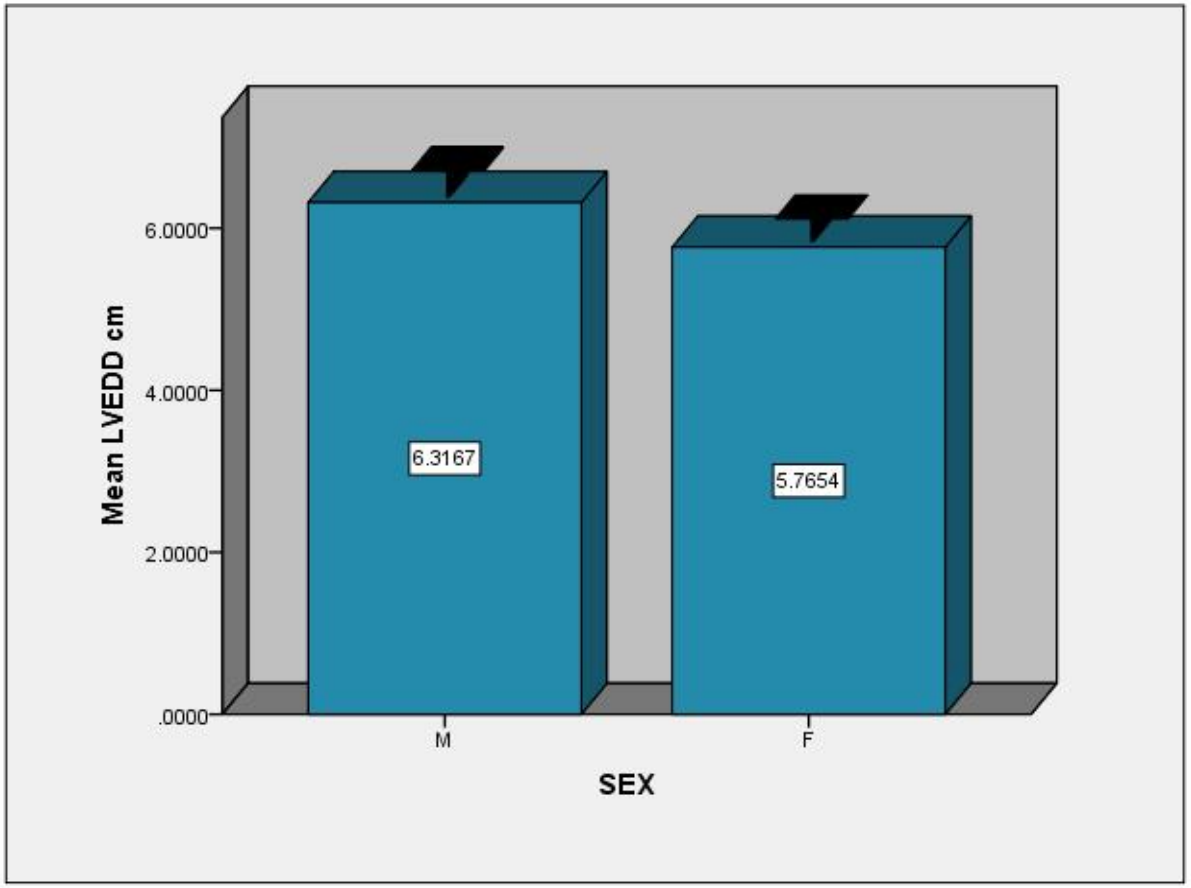
This table shows

1) Negative correlation of QRS duration with age.
2) Positive correlation of duration following myocardial infarction and QRS duration which is statistically significant.(p =0.001)
3) Negative correlation of QRS duration with left ventricular ejection fraction (LVEF),which is statistically significant(p<0.001)
4) Positive correlation of QRS duration with left ventricular end diastolic diameter,which is statistically significant(p=0.001)

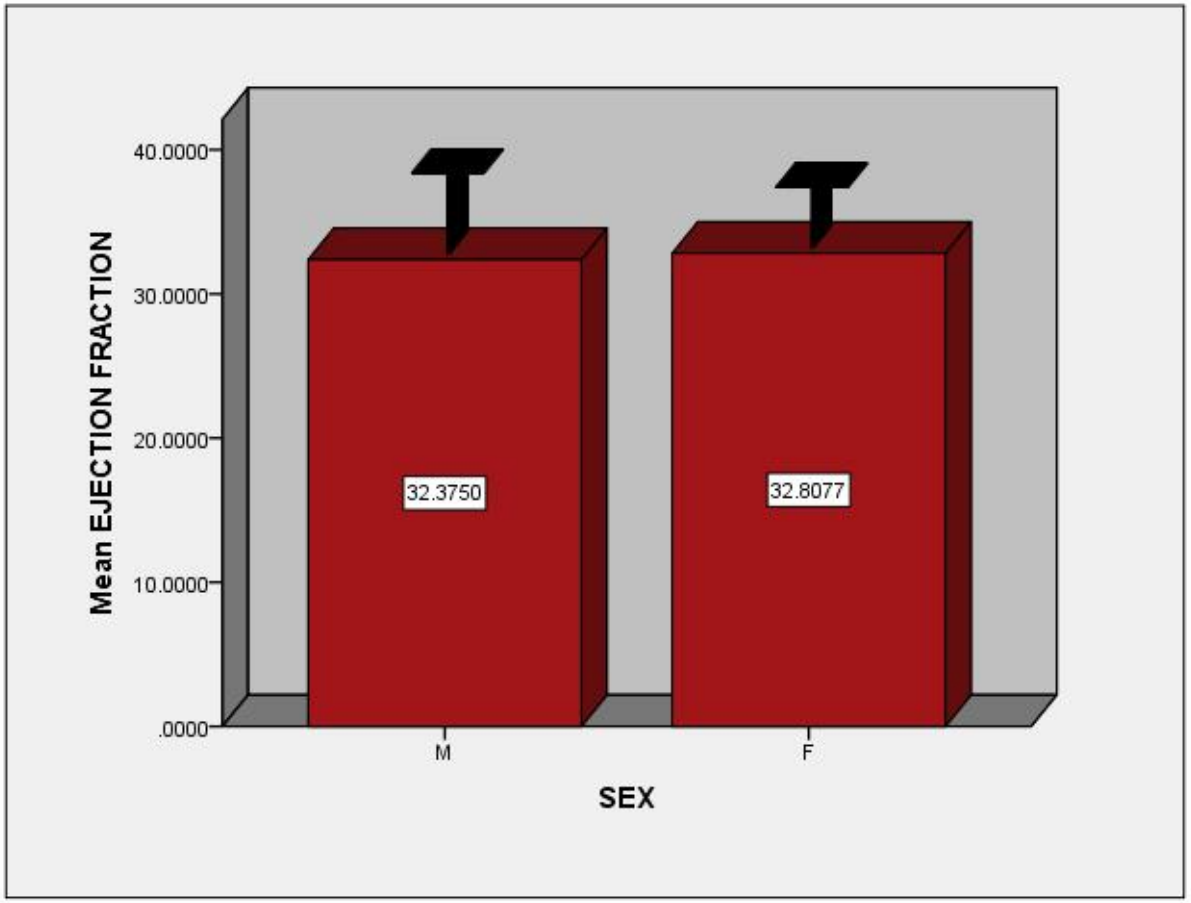
SEX DISTRUBUTION OF STUDY POPULATION



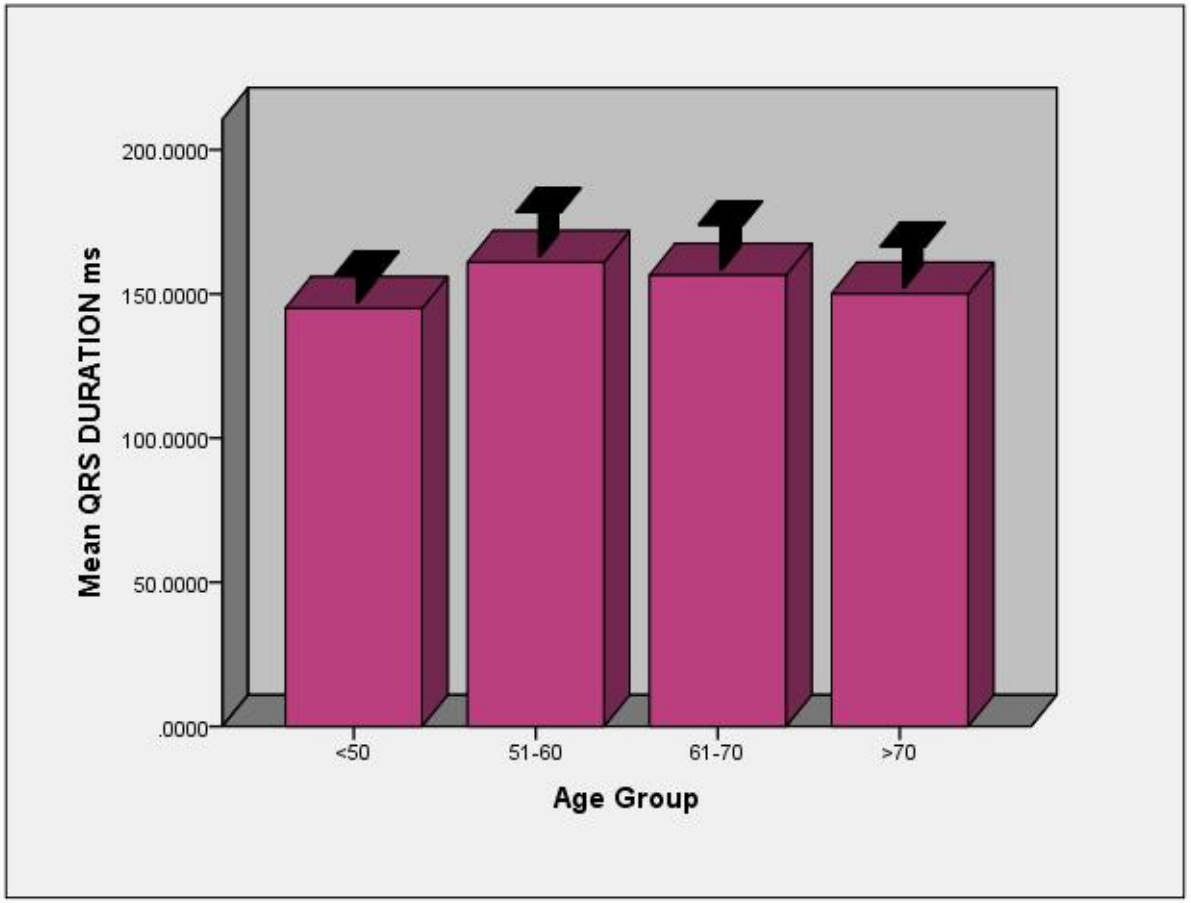
SEX DISTRIBUTION OF LVEDD



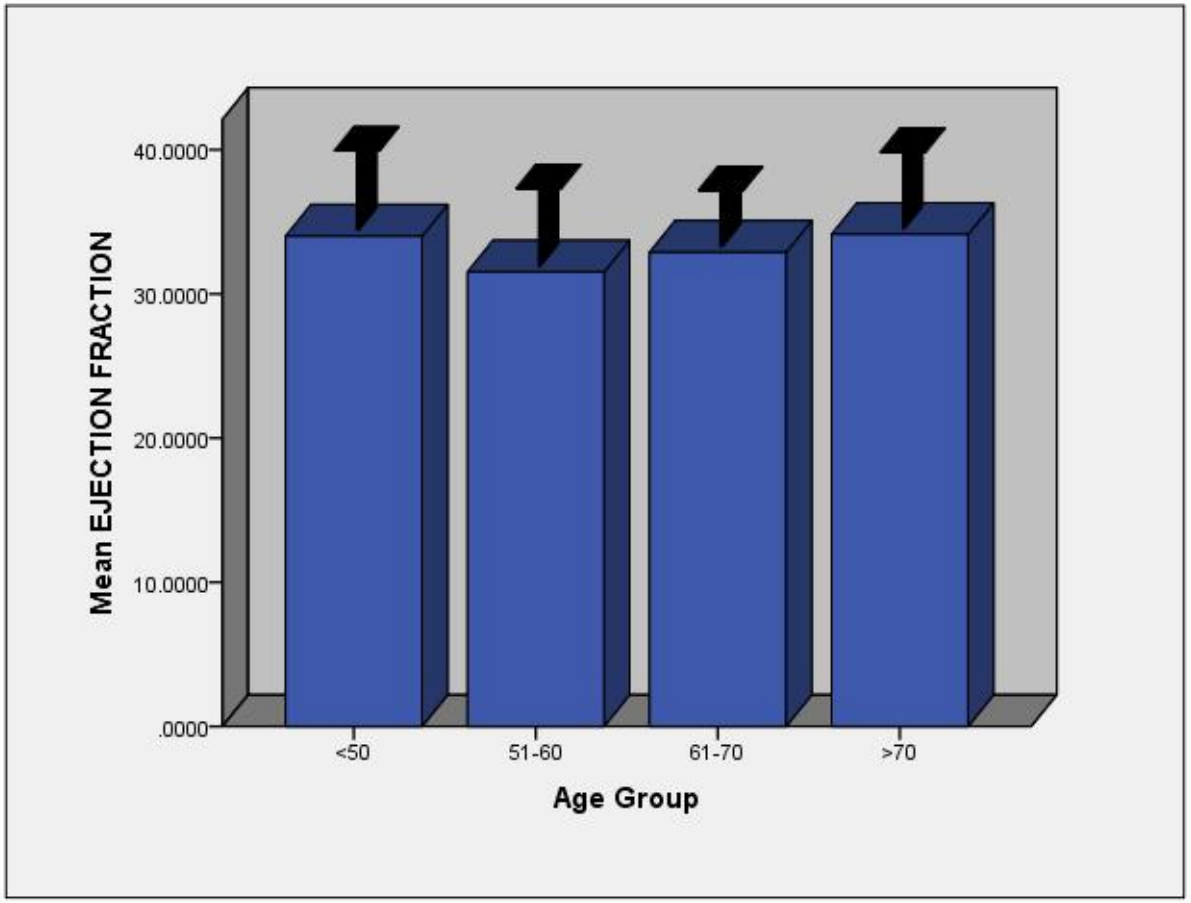
SEX DISTRIBUTION OF EJECTION FRACTION



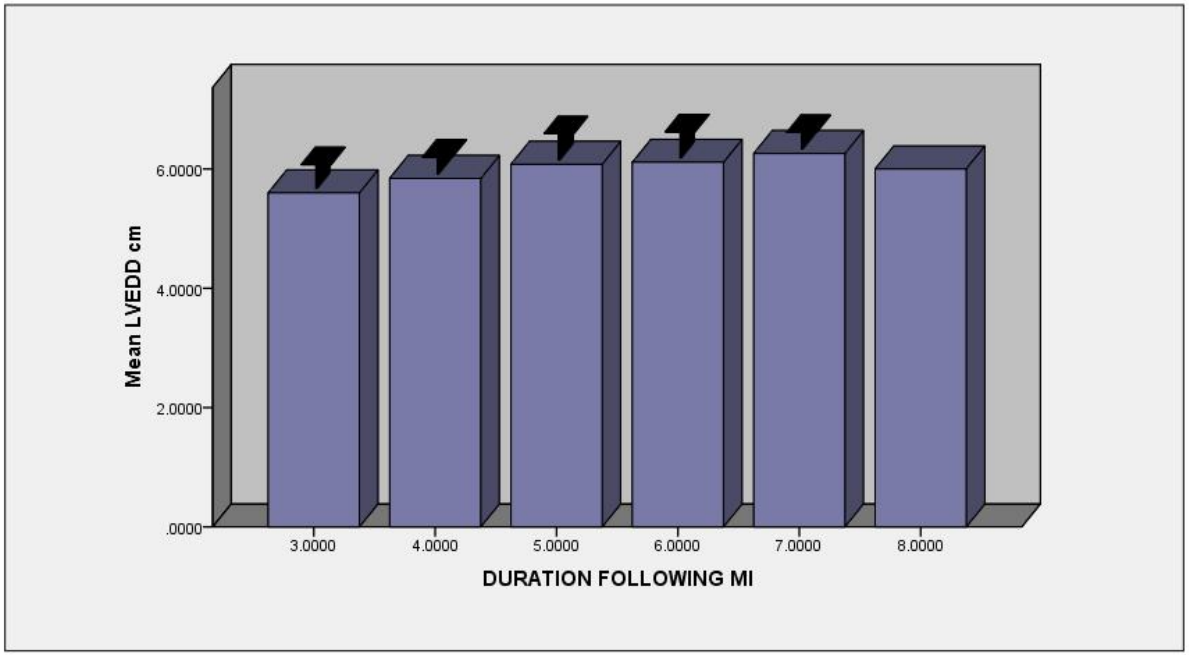
AGEWISE DISTRIBUTION OF QRS DURATION



AGEWISE DISTRIBUTION OF MEAN EJECTION FRACTION

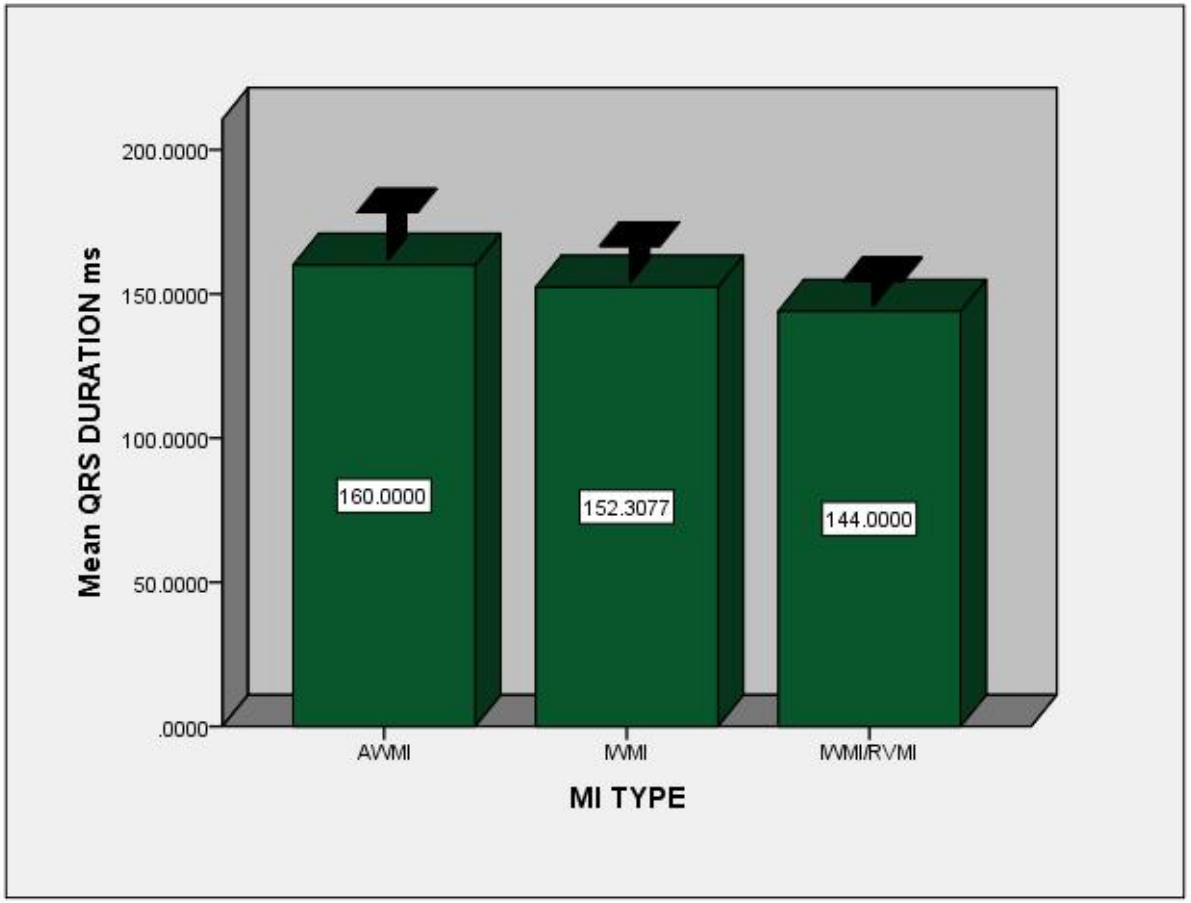


LVEDD RELATING TO DURATION FOLLWING MI



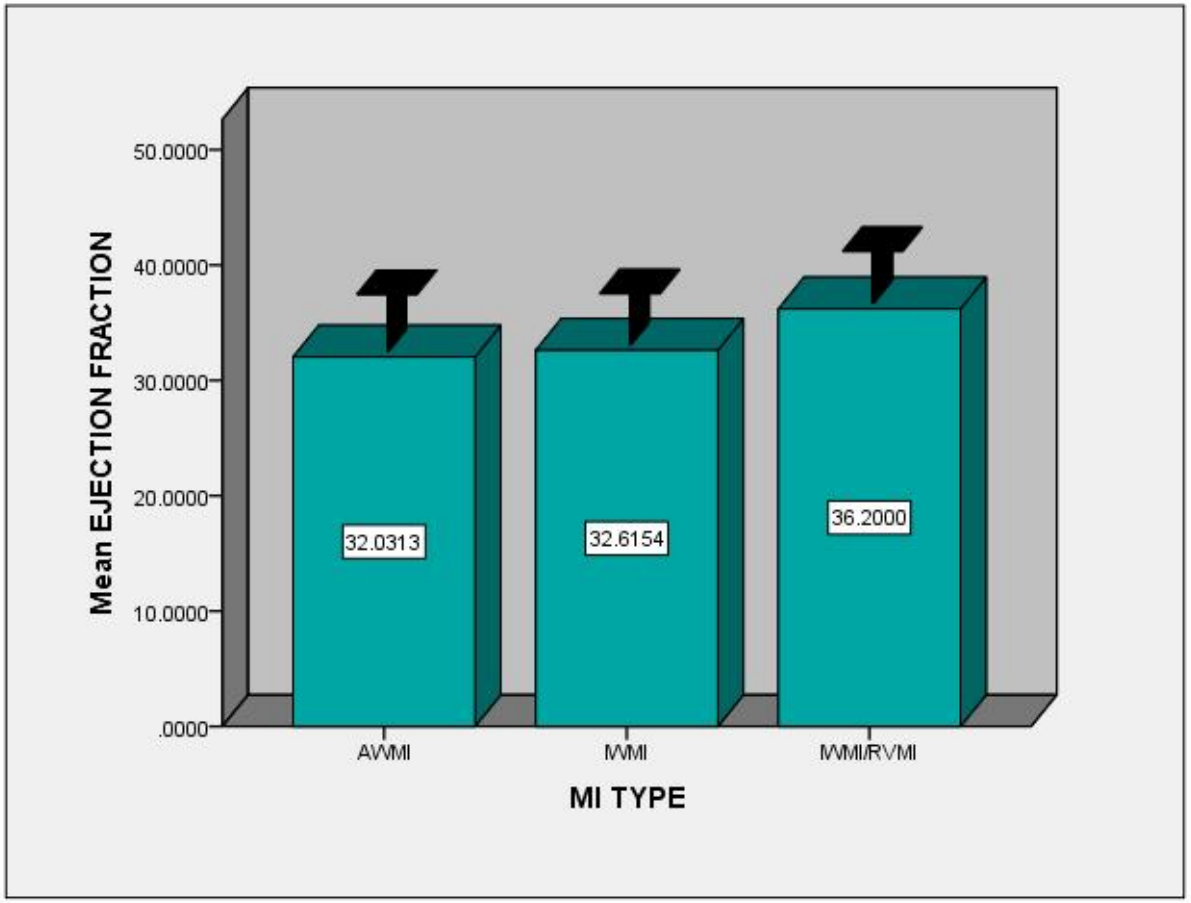
Error bars: +/- 1 SD

QRS DURATION RELATING TO MI TYPE

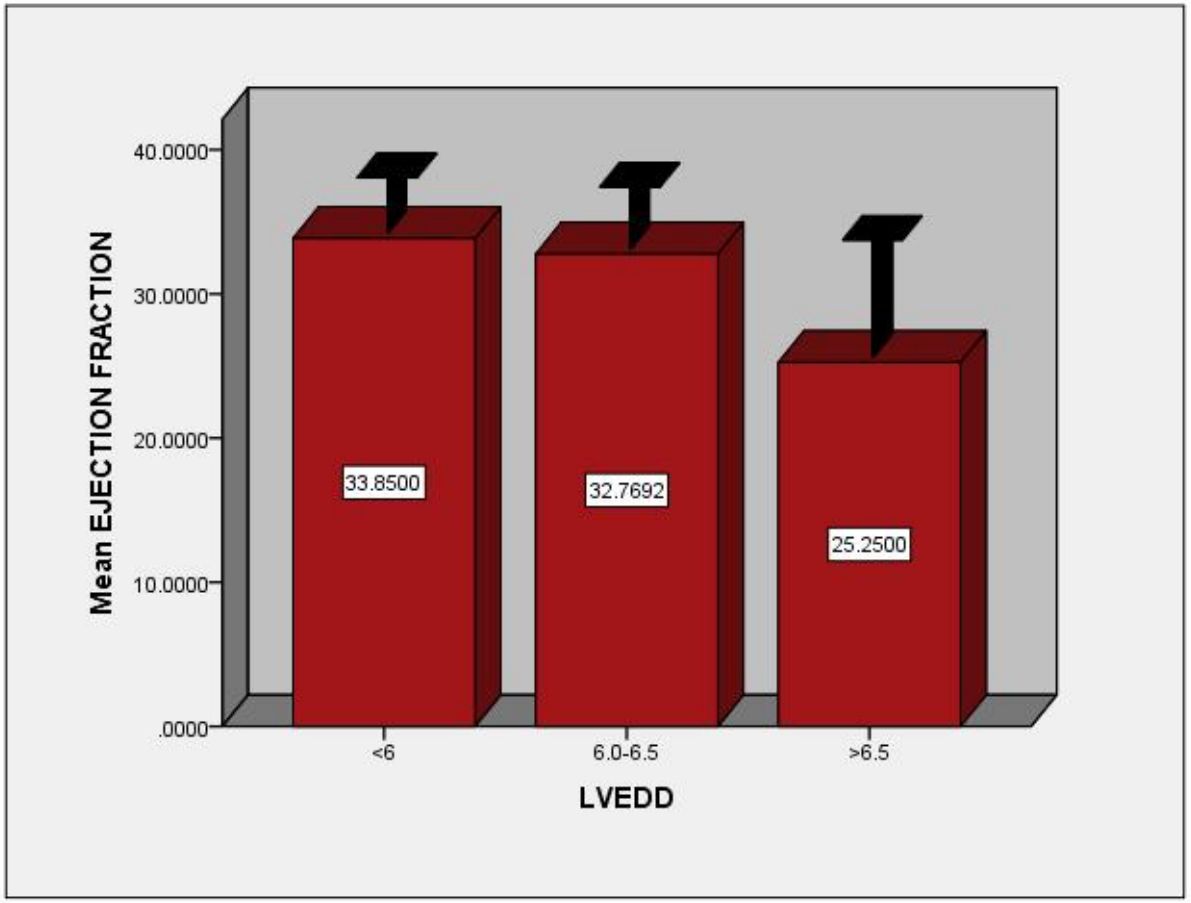


Error bars: +/- 1 SD

MEAN EJECTION FRACTION RELATING TO MI TYPE

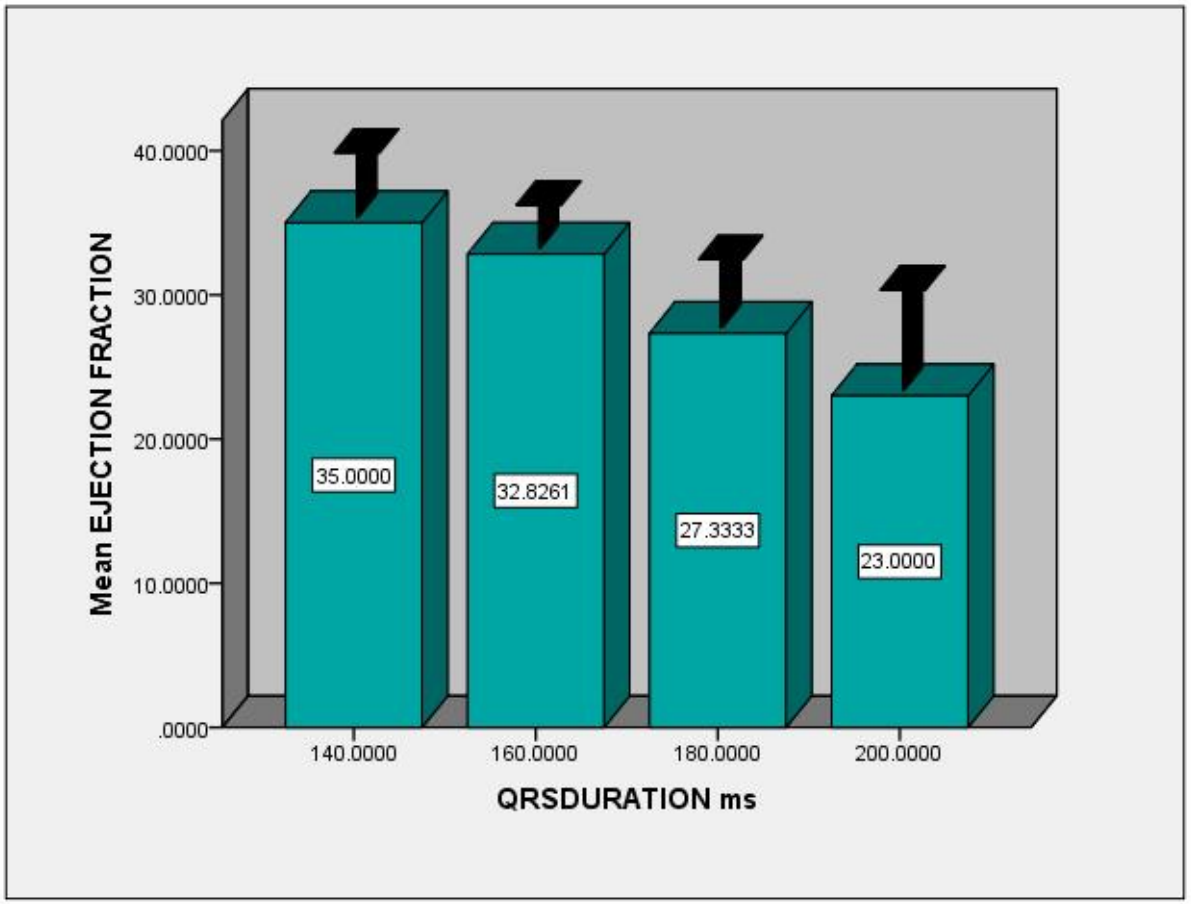


EJECTION FRACTION RELATING TO LVEDD



Error bars: +/- 1 SD

EJECTION FRACTION RELATING TO QRS DURATION



Error bars: +/- 1 SD

DISCUSSION

The term Ischemic cardiomyopathy is defined as a clinical syndrome occurring in coronary artery disease patients in which a lot of pathophysiological processes operate resulting in left ventricular dysfunction and heart failure symptoms. This condition is the predominant form of heart failure related to coronary artery disease. Burch et al. where the first to use the term Ischemic cardiomyopathy to describe a condition that produces clinical features often indistinguishable from that of primary dilated cardiomyopathy.

Conduction abnormalities in electrocardiogram of patients with ischemic cardiomyopathy include left bundle branch block, right bundle branch block, bi/tri-fascicular block, and nonspecific intra ventricular conduction defects. Such conduction abnormalities not only help to identify ischemic cardiomyopathy patients but also prognosticate them i.e. predict the mortality and morbidity of such patients.

According to Amir Kashani et al. prolongation of QRS ($\geq$120 ms) occurs in 14% to 47% of heart failure(HF) patients(26). Left bundle branch block is far more common than right bundle branch block. Left-sided intraventricular conduction delay is associated with more advanced myocardial disease, worse left ventricular (LV) function, poorer prognosis,

and a higher all-cause mortality rate compared with narrow QRS complex. It also predisposes heart failure patients to an increased risk of ventricular tachyarrhythmias, but the incidence of cardiac or sudden death remains unclear because of limited observations. A progressive increase in QRS duration is associated with worse prognosis.

Brenyo.A. et al. in their study found that QRS duration and morphology, evaluated via a standard 12-lead electrocardiogram (ECG), represent an opportunity to derive useful prognostic information regarding the risk of subsequent cardiac events or therapeutic outcomes(27). Prolonged QRS duration, and the presence of intraventricular conduction abnormalities, usually indicate the presence of changes in the myocardium due to underlying heart disease. Prolonged QRS duration is often associated with depressed ejection fraction or enlarged left ventricular volumes, but several studies have demonstrated that this simple ECG measure provides independent prognostic value, after adjusting for relevant clinical covariates. Post-infarction patients with prolonged QRS duration have a significantly increased risk of mortality. Prolonged QRS duration, and especially presence of left bundle branch block, seems to predict a benefit from cardiac resynchronization therapy in both ischemic and non-ischemic cardiomyopathy patients. Therefore, QRS duration and morphology should not only be considered a predictor of death or sudden

death in patients after myocardial infarction, and in those suspected of coronary artery disease, but also as a predictor of benefit from cardiac resynchronization therapy in patients with heart failure, whether of an ischemic or non-ischemic origin.

In a study by Kearney M.T.et al. found that in 184 patients with left ventricular ejection fraction < 35%, QRS duration was > 150 msin 53, and </= 150 ms in 131(28). They evaluated patients with baseline chest radiographs, echocardiograms, and Holter recordings. Patients with QRS duration above and below 150 ms were similar in age, sex, functional class, renal function, serum sodium, and ejection fraction. In patients with QRS > 150 ms, left ventricular end-diastolic and end-systolic diameters were greater than patients with QRS duration </=150 ms (P <.01). Patients with QRS > 150 ms had less low frequency R-R interval spectral power (P <.04). At 5 years 60% of patients with QRS > 150 ms had died compared with 35% of patients with QRS </=150 ms (P <.001). This increase in mortality was predominantly the result of an increase in progressive heart failure.

Huang X. et al. studied clinical, electrocardiographic and echocardiographic findings in 64 patients with dilated cardiomyopathy were retrospectively.(29).Compared with 51 patients without complete left

bundle branch block (CLBBB), 13 patients with CLBBB had higher New York Heart Association (NYHA) functional class ($P < 0.05$), increased left ventricular end-diastolic and end-systolic diameters ($P < 0.002$) and myocardial mass ($P < 0.02$), severe mitral regurgitation ($P < 0.01$) and higher mortality rate ($P < 0.04$). Multivariate stepwise regression analysis revealed that the presence of complete left bundle branch block was an independent prognostic factor for patients with dilated cardiomyopathy.

In our study, it has been established that as QRS duration increases, the left ventricular ejection fraction(LVEF) decreases and left ventricular end diastolic diameter increases (LVEDD).

CONCLUSION

In Ischemic cardiomyopathy patients

1) As the duration following Myocardial infarction increases, QRS duration also increases, as evidenced by Spearman's correlation coefficient of 0.443 & the association is statistically significant (p=0.001).

2) QRS duration shows a negative correlation with LV ejection fraction as evidenced Spearman's correlation coefficient – 0.532 i.e. as QRS duration increases,Left ventricular ejection fraction decreases & the association is statistically significant (p<0.001).

3) QRS duration shows a positive correlation with Left ventricular end diastolic diameter (LVEDD),as evidenced Spearman's correlation coefficient 0.439 , i.e. as QRS duration increases, the Left ventricular end diastolic diameter (LVEDD) also increases& the association is statistically significant (p=0.001).

4) The incidence of

   a) Left bundle branch block (LBBB)is 78%(52%+ 26%)

   b) Left bundle branch block (LBBB) + Left anterior fascicular block(LAFB) is 12%

   c) Right bundle branch block (RBBB) is 6%

   d) Right bundle branch block (RBBB) +Left anterior fascicular block(LAFB) is 4%

BIBLIOGRAPHY

1. An Introduction to Electrocardiography by Leo Schamroth -- Seventh Edition.,

2. Lipman—Massie Clinical Electrocardiography by Bernard S.Lipman M.D., Marvin I.Dunn M.D.,---Eighth edition.

3. JamesT.N:The connecting pathways between the sinus node and A.V node and between the right and left atrium in human heart. Am Heart Journal 1963;66:498

4. Hoffman BF and Cranefield PF: Electrophysiology of the heart New York,New York 1967

5. James T.N ,Sherf L., Fine structure of HIS bundle,Circulation 1971:44:9

6. Kulbertus HE &DemoulinJC;The conduction system anatomical and pathological aspects in Krekler DM, Goodwin JF: Cardiac arrhythmias: The modern electrophysiological approach.London WB Saunders Co p161975

7. SherAM:The sequence of ventricular excitation Am J of Cardiology 1964:14:287

8. James T.N:Anatomy of coronary arteries New York Paul B HobberInc 1961

9. DodgeHT,GrantRP:Mechanisms of QRS prolongations in man,Right ventricular conduction defects Am J Med 1956: 21:534 – 550

10. Hoffman B.F 1961 Problems in cardiac electrophysiology.In Biophysics of Physiological and Pharmacological actions,edShanes A.M Washington,DC:Am. Assoc.Adv.Sci

11. Rodriguez M.I&Sodi-PallaresD.1952.The mechanism of complete and incomplete bundle branch block. Am.Heart.J.44,715

12. Walston A.2,Boineauj.p.,Spach.M.S. et al 1968.Relationship between ventricular depolarization and QRS in right and left bundle branch block

13. Roenbaum M.B., Elizari M.V&Lazzari J.O (1970) The Hemiblocks. Oldsmar, Florida: Tampa Tracings.

14. Scamroth.L, Myburgh D.P, & Schamroth.C.L.,1985. The early signs of bundle branch block..Chest 87,180.

15. Sodi-PallaresD.,Medrano.G.,Bisteni a. & Ponce de Leon G.G. 1970. Deductive and polyparametric Electrocardiography.InstitutoNacional de Cardiologia de Mexico.

16. Fahr G. 1921 Some fundamental principles of electrocardiography. Arch. Intern. Med 27, 126 – 30

17. Moore E.N., Hoffman B.F., Patterson D.F.& Stuckey J.H 1964.Electrocardiographic changes due to delayed activation of the wall of the right ventricle

18. Roenbaum M.B., Elizari M.V&Lazzari J.O.,NauG.J.,Halpern M.S. & Levi R.J. 1972.The differential electrocardiographic manifestations of hemiblocks, bilateral bundle branch block and trifascicular blocks. In Advances in Electrocardiography ,ed. Schlant R c. & Hurst J.W., pp.149- 220 ,New York Grune& Stratton.

19. Johnson R.L, Averill K.H@ Lamb L.E 1960 Electrocardiographic findings in 67,375 asymptomatic induviduals. Part 6. Right bundle branch block Am.J.Cardiol

20. Scamroth.L,&(19640Bradlow.B.A.,Incomplete left bundle branch block.Br.Heart.J

21. Schneider J.,Thomas H.E., McNamara P.M., &Kannel W.B (1985). Clinical electrocardiographic study of newly acquired left bundle branch block: The Framingham study Am.J.Cardiol 55, 1332-8.

22. Schwartz D.B>&Schamroth L. (1979) .therffect of pregnancy on frontal plane QRS axis. J.Electrocardiol. 12, 279—81.

23. Roenbaum M.B., Elizari M.V., & Levi R.J., (1969). Five cases of intermittent left bundle branch block. Am.JCardiol. 24,1.

24. Rothfeld E.L, Zucker I.R.,& Tiu R.,(1969). The electrocardiographic syndrome of superior axis and right bundle branch block. Dis Chest, 55.306.

25. Medrano G.ABrenes P.C De Micheli A.&SodiPallares D.(1969). El boqueo de la subdivision anterior de la ramaizquierda solo o asociado al boqueo de la rama de racha. Aracha.InstCardiol. Mexico 39, 672

26. Amir Kashani, MS, MD., S. Serge Barold, MD,Significance of QRS Complex Duration in Patients With Heart Failure., J Am CollCardiol, 2005; 46:2183-2192.

27. Brenyo A., Zareba W., Prognostic significance of QRS duration and morphology.Cardiol J. 2011;18(1):8-17

28. Kearney M.T.,Zaman A.,Eckberg D.L., Lee A. J, Fox K.,Cardiac size, autonomic function, and 5-year follow-up of chronic heart failure patients with severe prolongation of ventricular activation.., J Card Fail. 2003 Apr;9(2):93-9.

29. Huang X., ShenW.,Gong L.,Clinical significance of complete left bundle branch block in dilated cardiomyopathy.Chin Med Sci J. 1995 Sep;10(3):158-60

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A study on conduction abnormalities and QRS duration in patients with ischemic cardiomyopathy and their correlation with LV ejection Fraction

Principal Investigator : Dr.S.Karthikeyan PG in MD(GM)

Designation : PG in MD(GM)

Department : Department of Medicine
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 18.04.2011 at the Modernized Seminar Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate form the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

PROFORMA

S.no:

Date:

Name:

Age:

Sex:

Address:

Contact phone no:

Occupation:

Presenting complaints

Past history of Diabetes/ Hypertension/Myocardial infarction:

Personal history: Alcoholism,smoking.

Pulse: BP:

CVS: RS:

ECG :(QRS, Types of conduction abnormalities)

ECHOCARDIOGRAPHY

Left Ventricular Ejection Fraction (LVEF):

Left ventricular end diastolic diameter (LVEDD):

Regional Wall motion abnormality:

S. NO	NAME	SEX	AGE	DM	HT	SMOKING	ALCOHOL	MITYPE	DURATION	QRSDURATION	BLOCK	EF	LVEDD
1	Mahesh.K	M	65	yes	yes	yes	yes	AWMI	4	140	LBBB	38	6
2	Muthu.T	M	55	yes	no	yes	yes	AWMI	6	160	LBBB	30	6.4
3	Kumar.D	M	73	yes	no	no	no	IWMI/RVMI	5	140	RBBB	39	6
4	Janaki.S	F	54	no	no	no	no	IWMI	6	180	LBBB	26	6
5	Jamuna.R	F	67	yes	no	no	no	AWMI	7	160	LBBB	35	5.8
6	Fardeen.H	F	48	no	yes	yes	yes	IWMI	4	140	LBBB	26	6
7	Gopal.T	M	56	no	yes	yes	yes	AWMI	6	160	LBBB	34	6.5
8	Navaneethan.L	M	74	yes	no	yes	yes	AWMI	5	180	LBBB+LAFB	22	6.9
9	Murthy.G	M	63	yes	yes	yes	yes	IWMI	8	140	LBBB	37	6
10	Rayar.K	M	67	no	yes	yes	yes	AWMI	7	200	LBBB+LAFB	28	6.5
11	Banu.R	F	69	yes	no	no	no	IWMI	5	140	LBBB	37	5.6
12	Mayi.S	F	55	no	yes	no	no	AWMI	4	140	LBBB	38	5.8
13	Jayanthi.S	F	65	yes	yes	no	no	AWMI	3	140	LBBB	26	5.3
14	Kala.D	F	76	yes	no	no	no	IWMI/RVMI	6	160	RBBB+LAFB	37	5.6
15	Jaya.Y	F	57	no	yes	no	no	AWMI	4	160	LBBB	40	5.9
16	Vidya.H	F	80	no	yes	no	no	AWMI	5	140	LBBB	32	6
17	Logu.J	M	67	no	yes	yes	yes	IWMI	6	160	LBBB	26	6.5
18	Hari.G	M	59	no	yes	yes	yes	IWMI	6	160	LBBB	30	5.6
19	Kamini.S	F	62	no	no	no	no	AWMI	4	160	LBBB	34	5.8
20	Ganesh.J	M	56	no	yes	yes	yes	AWMI	3	160	LBBB	38	5.9
21	Ragu.K	M	49	yes	no	yes	yes	IWMI/RVMI	7	140	RBBB+LAFB	38	6.1
22	Mukund.Y	M	72	yes	no	yes	yes	AWMI	4	140	LBBB	38	6
23	Mahesh.T	M	54	no	no	yes	yes	IWMI	7	160	LBBB	32	6.5
24	Asha.D	F	65	no	yes	no	no	AWMI	5	160	LBBB	30	5.2
25	Nithya.G	F	67	yes	no	no	no	AWMI	5	160	LBBB	34	6.3

S. NO.	NAME	SEX	AGE	DM	HT	SMOKING	ALCOHOL	MITYPE	DURATION	QRSDURATION	BLOCK	EF	LVEDD
26	Hameed.V	M	56	yes	no	yes	yes	AWMI	6	180	LBBB+LAFB	24	6.8
27	Yamini.H	F	83	yes	no	no	no	IWMI	6	160	LBBB	34	5.9
28	Selvi.D	F	49	no	yes	no	no	AWMI	4	140	LBBB	38	5.4
29	Ravi.A	M	59	no	yes	no	no	AWMI	5	160	LBBB	32	6.4
30	Paramasivam.A	M	58	no	no	yes	yes	IWMI	5	140	LBBB	39	6
31	Gayathri.K	F	56	yes	yes	no	no	AWMI	6	180	LBBB+LAFB	28	5.6
32	Ramammal.T	F	65	no	yes	no	no	AWMI	6	160	LBBB	31	6
33	Bala.R	M	69	yes	no	no	yes	AWMI	5	140	LBBB	37	6.6
34	Jaya.G	F	62	no	yes	no	no	IWMI	4	140	LBBB	36	5.5
35	Kuppammal.K	F	58	yes	no	no	no	IWMI/RVMI	5	140	RBBB	39	6.2
36	Tamil.N	M	52	no	yes	no	no	AWMI	5	160	LBBB	35	6.1
37	Karuppiah.J	M	64	yes	yes	yes	no	AWMI	6	180	LBBB+LAFB	36	6.4
38	Dayalammal.v	F	72	yes	no	no	no	AWMI	4	140	LBBB	34	5.4
39	Veera.G	M	58	no	yes	no	yes	IWMI	7	160	LBBB	32	6.4
40	Jayabal.k	M	55	no	yes	yes	yes	AWMI	6	200	LBBB+LAFB	18	7
41	Uma.J	F	66	yes	no	no	no	IWMI	6	160	LBBB	32	5.8
42	Padma.K	F	70	yes	no	no	no	AWMI	4	160	LBBB	34	5.8
43	Vimala.K	F	58	no	yes	no	no	AWMI	5	160	LBBB	31	5.9
44	Chitra.G	F	54	no	no	no	no	AWMI	4	140	LBBB	28	6
45	Isakki.D	F	73	yes	yes	no	no	IWMI	6	140	LBBB	37	5.4
46	Karthik.k	M	66	yes	no	yes	no	AWMI	5	160	LBBB	28	6.5
47	Ganesh.T	M	55	yes	yes	yes	yes	AWMI	4	160	LBBB	32	6.5
48	Booma.S	F	58	no	yes	no	no	AWMI	6	180	LBBB	28	6.3
49	Thillai.S	F	54	no	no	no	no	IWMI/RVMI	5	140	RBBB	28	5.4
50	Ramu.J	M	50	no	no	yes	yes	AWMI	6	160	LBBB	34	6